BUCKEYE ROT OF TOMATO IN CALIFORNIA[1]

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INTRODUCTION

Although buckeye rot of green tomato fruits (*Lycopersicum esculentum* Mill. var. *vulgare* Bailey), caused by *Phytophthora terrestria*, was described by Sherbakoff (*25*) [2] more than two decades ago, the disease was not known to occur in California, where tomatoes are a major crop, until September 1937 when it was observed by J. B. Kendrick in a 100-acre tomato field near Davis. The rot was confined to fruits on or near the ground following a heavy irrigation. In August 1938, infected green Italian pear-shaped tomatoes were received from Modesto, and the disease, based on symptoms only, was identified as buckeye rot. Two months later, this disease was found to be of major importance in the late crop of green-wrap tomatoes grown at Brentwood.

Buckeye rot of green tomato fruits has consistently been ascribed to *Phytophthora parasitica* by investigators in various states and foreign countries. Some of the reported diagnoses were made, apparently, on symptomatology alone or at least on very limited cultural tests. However, Bewley (*4*), in England, may have been the first to suggest that more than one species of *Phytophthora* may cause buckeye rot, naming *P. cryptogea* in addition to *P. parasitica*. Investigations in California not only support Bewley's contentions, but, in addition, indicate that the disease may be produced by other species in the absence of *P. parasitica*.

DISTRIBUTION OF THE DISEASE

According to Sherbakoff (*25*, *26*, *27*), buckeye rot of tomato fruits was first recognized as a new disease in January 1915 at Goulds, Fla., but presumably had been observed as early as 1911, and occurs along both the east and west coasts. The disease has been reported to the Division of Mycology and Disease Survey, United States Department of Agriculture,[3] from Arizona, Arkansas, Colorado, Florida, Illinois, Indiana, Louisiana, Maryland, Massachusetts, Mississippi, Missouri, New Jersey, New York, North Carolina, Ohio, Tennessee, Texas, Virginia, and West Virginia. It also occurs in Puerto Rico. The first record of the disease from California was given by Ramsey [4] in 1939. In view of its wide geographical range, Weimer (*39*) was undoubtedly correct in stating that "buckeye rot of tomatoes, then, is not restricted to the extreme southern States."

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[1] Received for publication August 14, 1940. Joint contribution from the Division of Plant Pathology, California Agricultural Experiment Station, and the Department of Botany, Missouri Agricultural Experiment Station (Journal Series No. 687).

[2] Italic numbers in parentheses refer to Literature Cited, p. 472.

[3] Letter from H. A. Edson, Division of Mycology and Disease Survey, U. S. Department of Agriculture, dated February 19, 1940.

[4] RAMSEY, G. B. FRUIT AND VEGETABLE DISEASES ON THE CHICAGO MARKET IN 1938. U. S. Dept. Agr. Bur. Plant Indus., Plant Disease Rptr., Sup. 114, pp. [27]–40. 1939. [Mimeographed.]

The disease has also been reported in Argentina (*5*), Australia (*14*), the British West Indies (Trinidad, St. Vincent, and Montserrat) (*1*), Canada (*7*), Denmark (*10, 37*), England (*2, 3, 4, 15, 16, 17*), India (*33*), Japan (*29*), Mexico (*28*), the Netherlands (*18*), Palestine (*20*), and the Union of South Africa (*34, 35*).

Further records, which may possibly relate to buckeye rot, occur in the literature and are briefly mentioned in order to make this paper as inclusive as possible. A rot of green tomato fruits, presumably caused by *Phytophthora terrestria*, was found in Cuba in 1918 by Bruner, according to Weimer (*39*). In 1932, just 7 years prior to the first definite record of the disease in the British West Indies (*1*), Briant (*6*) found that an unnamed species of *Phytophthora* was responsible for a fruit rot of tomatoes in Trinidad. In Bermuda, a species of *Phytophthora* belonging to the *P. palmivora* group, was believed by Waterston (*36*) to be the cause of a rot of green tomato fruits (varieties Marglobe, Pritchard, and Break o'Day).

Although the disease has not been reported from Greece and Italy, nevertheless it is significant that Sarejanni (*24*) and Goidanich (*9*) have recently described a collar rot of tomato plants caused by *Phytophthora parasitica*. Samuel (*22*) stated the disease has never been observed in South Australia, although the disease is known to occur in the metropolitan area of Sydney, Australia (*14*).

A rot of tomato fruits, caused by *Phytophthora capsici*, has occurred in the Arkansas Valley of Colorado (*23*), but whether the disease is identical with buckeye rot was not indicated. [5]

SYMPTOMS OF THE DISEASE

The symptoms of buckeye rot of green tomato fruits collected in the Brentwood and Stockton sections, two of the four known centers of infestation in California, are identical with the original description as given by Sherbakoff (*25, 26, 27*) and later by Kendrick (*11*), Ramsey and Link (*19*) whose paper is illustrated by excellent colored photographs, Wager (*34, 35*), Weber and Ramsey (*38*), and Young, Harrison, and Altstatt (*40*). Green fruits of all sizes, if in contact with or near the surface of moist or waterlogged soil, were subject to infection. Irregular-shaped, water-soaked areas, usually but not always at the blossom end of the fruits, constituted the first visible evidence of infection (fig. 1, *A*). As the lesions enlarged, the centers turned brown to blackish-brown and frequently developed either complete or incomplete concentric brown rings (fig. 1, *B, D*). Some fruits were devoid of this ring symptom. On all lesions, however, the advancing margin was water-soaked, while the rate of enlargement was rapid under conditions of high temperature and humidity and an excessive supply of irrigation water. Decayed areas were firm in texture, with little or no aerial mycelium on the surface of the fruits. An abundant mycelial growth developed, however, when diseased fruits were placed in a moist chamber, and the rate of decomposition was accelerated. Lesions on some fruits showed arrested development, the surface tissues becoming hard and dry (fig. 1, *C*).

[5] Since this paper was accepted for publication the following article has appeared: KREUTZER, W. A., BODINE, W. W., and DURRELL, L. W. CUCURBIT DISEASES AND ROT OF TOMATO FRUIT CAUSED BY PHYTOPHTHORA CAPSICI. Phytopathology 30: 972-976, illus. 1940. In this article a field decay of tomato fruits, caused by *Phytophthora capsici*, is described in more detail.

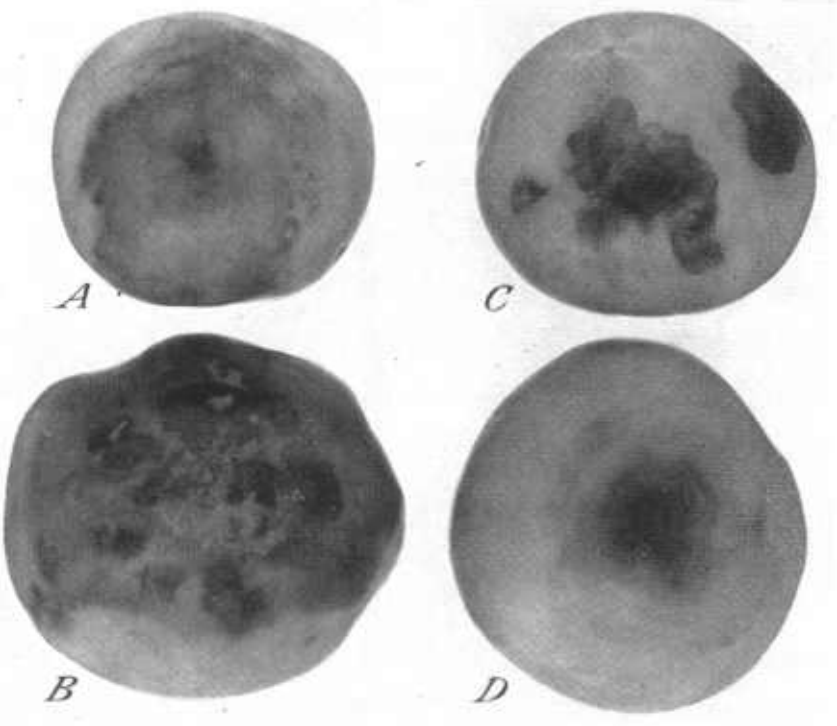

FIGURE 1.—Natural infection of green-wrap tomato fruits grown at Brentwood, Calif.: *A*, Large, irregular-shaped, water-soaked lesion, with brown center; *B*, an aggregate of brown lesions surrounded by a water-soaked margin; *C*, blackish-brown lesions showing arrested development; *D*, water-soaked lesion with brown center and concentric rings.

THE CAUSAL FUNGI, PHYTOPHTHORA CAPSICI AND P. DRECHSLERI

Small tissue fragments were removed aseptically from the advancing, internal margin of lesions of naturally infected, green tomato fruits, planted on malt-extract agar (*13*) in Petri dishes, and incubated at room temperature. Usually, after 48 hours, pure cultures were established on agar slants by transferring hyphal tips from the edge of the colonies. Cultures were made from approximately 300 green tomato fruits in 1938 and 1939.

Although the original cause of buckeye rot was ascribed by Sherbakoff (*25*) to *Phytophthora terrestria*, Tucker (*32*) later determined that the fungus was identical with *P. parasitica* Dast.

Microscopic examination of the California isolates indicated that two species of *Phytophthora* were involved in the rotting of green tomato fruits, namely, *P. capsici* Leonian (*12*) and *P. drechsleri* Tucker (*30*).[6] Cultures of *P. drechsleri* were more numerous than *P. capsici* in 1938, whereas in 1939 all the isolates were referable to *P. capsici*.

[6] An isolate from a rotting tomato fruit collected at Edison, Calif., in November 1940 was identified as *Phytophthora parasitica* Dast. The isolate developed the tufted type of mycelial growth characteristic of *P. parasitica*, formed sporangia and chlamydospores in culture, and exhibited temperature growth relations typical of the species. The isolate was indistinguishable from cultures obtained from tomato fruits from Cuba, Florida, and Missouri.

It is significant that not a single isolate of *P. parasitica* appeared during either season, although this species has heretofore been recognized as the exclusive causal agent of the disease.

The three species have similar temperature-growth relations and may be expected to appear on tomato fruits under similar environmental conditions. *Phytophthora drechsleri* is readily distinguishable from the other species by its nonpapillate sporangia which usually develop sparingly, even when hyphae are transferred to sterile distilled water. *P. capsici* does not develop typical chlamydospores and may thus be distinguished from *P. parasitica*. Furthermore, the sporangia of *P. capsici* are usually somewhat irregular, with a tendency to elongate in the apical region, while those of *P. parasitica* are more frequently of the regular, limoniform type. All three species are variable in production of oogonia, some isolates developing them in fair numbers while in others the sexual stage is never observed.

Tucker (*32*) has shown that the ability to invade and kill stems of pepper plants is a specific character of *Phytophthora capsici*. All isolates from tomato fruits that resembled *P. capsici* morphologically were inoculated into pepper stems; in every instance the fungus invaded and killed the terminal 3 to 6 inches of the stem in 5 days.

Pure cultures of *Phytophthora capsici* and *P. drechsleri*, isolated from naturally infected green tomato fruits collected at Brentwood, Calif., and of *P. parasitica* from an infected tomato fruit collected at Columbia, Mo., were used in the infection experiments. The fungi were grown on malt-extract agar in Petri dishes, incubated at room temperature, and used for inoculum when 4 days old.

Parallel inoculations were made on unwounded, detached, green tomato fruits in the laboratory and on green fruits in situ on healthy tomato plants grown in the greenhouse. In the laboratory, healthy fruits were washed in running tap water, rinsed in distilled water, and dried. A small block of inoculum was placed on the uninjured epidermis and kept moist with absorbent cotton under an inverted preparation dish. In some instances, inoculated fruits were placed in glass moist chambers. No fruits were wounded in any of the inoculation tests. For controls, fruits were handled in the same manner except that sterile agar was substituted for the inoculum.

In the greenhouse, the green fruits on living tomato plants, varieties Sutton's Best of All and Crackerjack, were not washed. The inoculum and moist absorbent cotton were held in place with a rubber band. One to several inoculated fruits were enclosed in a glassine bag after inoculation. A total of 12 fruits were inoculated with each species of *Phytophthora*. Control fruits on living plants were similarly treated except that sterile agar was substituted for the inoculum. All inoculated fruits became diseased, while the noninoculated control fruits remained healthy. The average incubation period was: *Phytophthora capsici*, 7 days; *P. drechsleri*, 11 days; and *P. parasitica*, 7½ days. These artificially induced lesions were identical in color and consistency with those resulting from natural infection. Concentric rings, either complete or incomplete, developed in some lesions (fig. 2, *A*), but usually this symptom was lacking (fig. 2, *B*). It is doubtful whether the zonation said to be characteristic of buckeye rot is a good distinguishing character. In these inoculation tests, it was observed that fruits vary a great deal in the development of the zonate

character, even when infected by the same isolate. The 3 species of *Phytophthora* produced identical symptoms when compared with each other, and it was impossible, therefore, to differentiate between them by using expressed symptoms as criteria. Laboratory tests yielded comparable results, with no essential difference in the incubation periods of the respective species tested. Reisolations were made from all infected fruits, and infection was again obtained with the reisolates.

These tests for pathogenicity indicate that species other than *Phytophthora parasitica* may also cause buckeye rot of tomato, and, in California, they are represented by *P. capsici* and *P. drechsleri*.

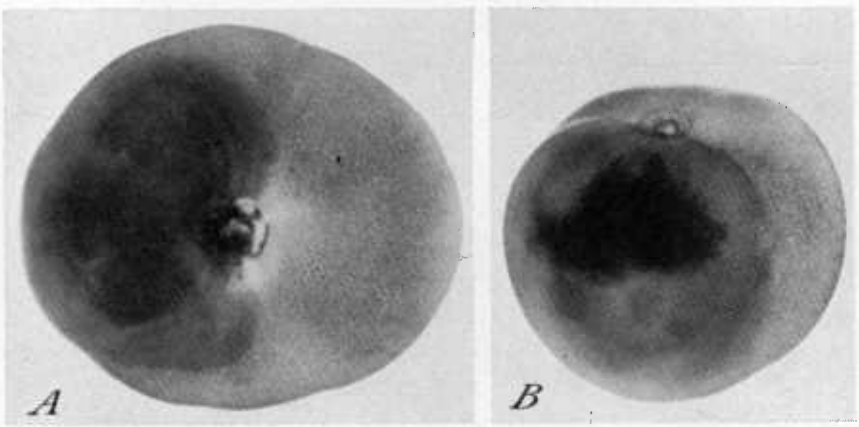

FIGURE 2.—Artificial infection, without wounding, of green Crackerjack variety tomato fruits, in situ on plants grown in the greenhouse. *A*, Large, water-soaked lesion, with slightly darker center, showing concentric ring effect produced by *Phytophthora capsici; B*, large, water-soaked lesion, with blackish-brown center, produced by *P. drechsleri*.

An isolate of *P. capsici* from a naturally infected honeydew melon fruit (*Cucumis melo* L. var. *inodorus* Naud.) has previously been shown to be pathogenic to green tomato fruits (*31*), resulting in lesions typical of buckeye rot.

That green or immature tomato fruits are highly susceptible and may be invaded by *Phytophthora parasitica* in the absence of wounds was previously mentioned by Sherbakoff (*25*), Gardner (*8*), Kendrick (*11*), Ramsey and Link (*19*), Rosenbaum (*21*), Wager (*35*), and Weber and Ramsey (*38*), so that the results presented in this paper are merely confirmatory.

DIFFERENTIAL HOSTS

Parallel inoculations of various fruits, roots, and tubers were made in the laboratory with 4-day-old cultures of *Phytophthora capsici*, *P. drechsleri*, and *P. parasitica* in an attempt to find a differential host which could be used for rapid identification of various isolates from naturally infected green tomato fruits. Infection of ripe tomato, eggplant (*Solanum melongena* L. var. *esculentum* Nees), avocado (*Persea gratissima* Gaertn.), Bartlett pear (*Pyrus communis* L.), and honeydew melon fruits was obtained with the three species of *Phytophthora*, but all produced identical symptoms on these hosts.

However, Yellow Crookneck and Zucchini pumpkin fruits (*Cucurbita pepo* L. var. *condensa* Bailey) were readily infected by *Phytophthora capsici* and *P. parasitica*, but not by *P. drechsleri;* Purple Top White Globe turnip roots (*Brassica rapa* L.) were infected only by *P. drechsleri* and *P. parasitica;* and carrot roots (*Daucus carota* L. var. *sativa* DC.) by *P. capsici* and *P. drechsleri. P. capsici* alone caused infection of bell or sweet green pepper (*Capsicum annuum* L. var. *grossum* Sendt.), Newtown Pippin apple (*Pyrus malus* L.), and cucumber fruits (*Cucumis sativus* L.). Thus, for the isolates tested, pumpkin and bell pepper fruits and turnip and carrot roots may be used as differential hosts.

All three species of *Phytophthora* failed to infect Garnet Chili and White Rose potato tubers (*Solanum tuberosum* L.); muskmelon (*Cucumis melo* L.), Klondike watermelon (*Citrullus vulgaris* Schrad.), and lemon fruits (*Citrus limonia* Osbeck); and garden beet roots (*Beta vulgaris* L. var. *crassa* Alef.).

SUMMARY

Buckeye rot of green tomato fruits is prevalent in several of the interior valleys of central California.

The disease is favored by high temperatures and humidity, and the most important factor favoring infection in California appears to be contact of the fruit with moist soil or with irrigation water.

Isolation, inoculation, and taxonomic studies show that the disease in California is caused by *Phytophthora capsici* and *P. drechsleri*, rather than by *P. parasitica.*

Several differential hosts, including pumpkin and bell pepper fruits and carrot and turnip roots, were found. These may assist in the identification of *Phytophthora* species isolated from diseased tomato fruits.

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